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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
GAMBEL, PHILLIP	
ART UNIT	PAPER NUMBER
1644	89

DATE MAILED: 08/20/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	08/349479	BORDON	
	Examiner	Art Unit	
	GAMBEL	1644	

– The MAILING DATE of this communication appears on the cover sheet with the correspondence address –

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☑ Responsive to communication(s) filed on 2/25/03 AND 8/12/03

2a)☐ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☑ Claim(s) _____ is/are pending in the application. 21, 35

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☑ Claim(s) _____ is/are rejected. 21 35

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a)☑ The translation of the foreign language provisional application has been received.

15)☑ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☑ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____.
4) ☑ Interview Summary (PTO-413) Paper No(s). _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other:

DETAILED ACTION

1. The Board of Patent Appeals and Interferences' Decision on Appeal NO.: 2002-2285, mailed 12/19/03 (Paper No. 85), is acknowledged.

2. Applicant's amendment, filed 2/25/03 (Paper No. 86), has been entered.
Claims 22, 23 and 25 have been canceled.

Claims 1-10, 13-20, 24, 26-34 have been canceled previously.

Applicant's supplement amendment, filed 8/12/03 (Paper No. 88), has been entered.
Claim 35 has been entered.

Given applicant's supplement amendment, filed 8/12/03 (Paper No. 88),
claims 11-12 have been canceled.

Claims 21 and 35 are currently and being acted upon presently.

3. Upon consideration of applicant's amended and canceled claims, the previous rejection under 35 U.S.C. § 102(e) as being anticipated by Dasch et al. (U.S. Patent No. 5,772,998) as it would read on the instant claims has been withdrawn.

4. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office Action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(f) or (g) prior art under 35 U.S.C. 103(a).

5. Claim 21 stands rejected under 35 U.S.C. § 103(a) as being unpatentable over Dasch et al. (U.S. Patent No. 5,772,998) in view of Ruoslahti et al. (U.S. Patent No. 5,583,103) AND/OR Bassols et al. (J. Biol. Chem. 263: 3039-3045, 1988).

Dasch et al. teach the use of TGF-β-specific antibodies to neutralize the effects of TGF-β, including lung fibrosis, liver cirrhosis fibrotic skin disorders and scarring (see entire document, including columns 5-6 and the Claims). Dasch et al. differs from the claimed methods by not disclosing that TGF-β was responsible, at least in part, for glomerulonephritis.

Ruoslahti et al. teach that it was known that excessive accumulation of extracellular matrix in glomerulonephritis was a diseases with a detrimental involvement of TGF-β (see column 2, paragraph 1) and that by treating TGF-β regulated activities, one treats certain pathologies including fibrotic disease and glomerulonephritis (see columns 5-6, overlapping paragraph). Further, Ruoslahti et al. teach that TGF-β -specific antibodies were able to inhibit the activity of TGF-β (see column 13)

Bassols et al. teach TGF-β regulates the expression of the extracellular matrix chondroitin/dermatan sulfate proteoglycans (see entire document, including Abstract, pages 3041 and 3043). Also, Bassols et al. teach that TGF-β regulates proteoglycans in kidney and lung and that TGFβ induces kidney fibroblast proliferation (see pages 3040-3041).

Given the teachings of Dasch et al. that TGF-β-specific antibodies could neutralize the effects of TGF-β in a several disorders; one of ordinary skill in the art at the time the invention was made would have motivated to apply such TGF-β-specific antibodies in other disorders where TGF-β- played a role such as glomerulonephritis, as taught and indicated by Ruoslahti et al. and Bassols et al. From the teachings of the references, it was apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was prima facie obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary.

6. Claim 35 is rejected under 35 U.S.C. § 103(a) as being unpatentable over Dasch et al. (U.S. Patent No. 5,772,998) in view of Bassols et al. (J. Biol. Chem. 263: 3039-3045, 1988) and Raghu et al. (Am Rev Respir Dis 131: 281-29 (1985).

Dasch et al. teach the use of TGF-β-specific antibodies to neutralize the effects of TGF-β, including various fibrotic conditions such as lung fibrosis (see entire document, including columns 5-6 and the Claims). Although Dasch et al. teach treating interstitial lung fibrosis, Dasch et al. differs from the claimed methods by not disclosing targeting ARDS per se.

Bassols et al. teach TGF-β regulates the expression of the extracellular matrix chondroitin/dermatan sulfate proteoglycans (see entire document, including Abstract, pages 3041 and 3043), including that TGF-β regulates proteoglycans in lung epithelial cell and fibroblast proliferation (see pages 3040-3041).

In reviewing the role of the extracellular matrix in the pathogenesis of interstitial pulmonary fibrosis, Raghu et al. teach that interstitial fibrosis was a characteristic feature of ARDS (see entire document, including Introduction and Summary on page 281).

Given the teachings of Dasch et al. that TGF-β-specific antibodies could neutralize the effects of TGF-β in a several disorders; one of ordinary skill in the art at the time the invention was made would have motivated to apply such TGF-β-specific antibodies in other fibrotic disorders, such as interstitial lung fibrosis (see Dasch et al.) and ARDS, wherein interstitial fibrosis is a characteristic feature (see Raghu et al.). Similarly, given the role of the extracellular matrix in interstitial lung fibrosis and ARDS (see Dasch et al. and Raghu et al.) and the ability of TGF-β to stimulate lung epithelial cell and fibroblast proliferation, one of ordinary skill at the time the invention was made would have had a reasonable expectation that neutralizing TGF-β with TGF-β- specific antibodies would be effective in treating fibrotic diseases, including ARDS. From the teachings of the references, it was apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was prima facie obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary.

7. Claims 21 and 35 are rejected under 35 U.S.C. § 103(a) as being unpatentable over
Dasch et al. (U.S. Patent No. 5,772,998) in view of Ruoslahti et al. (U.S. Patent No. 5,583,103) AND/OR Bassols et al. (J. Biol. Chem. 263: 3039-3045, 1988) for the reasons above set forth in Section 4 as it applies to claim 21
AND/OR
over Dasch et al. (U.S. Patent No. 5,772,998) in view of Bassols et al. (J. Biol. Chem. 263: 3039-3045, 1988) and Raghu et al. (Am Rev Respir Dis 131: 281-29 (1985) for the reasons above set forth in Section 5 as it applies to claim 35
and in further evidence of applicant's admission that given a generic method of decreasing TGF-β-induced production and deleterious accumulation of extracellular matrix associated with a pathology or a condition would render the specific fibrotic disorders such as glomerulonephritis, ARDS, cirrhosis or the liver and scarring as obvious species of one another (see Brief on Appeal, particularly Regarding Species Claims 23 and 25 and Regarding Genus Claim 21 on pages 18-24 of the Brief on Appeal, filed 3/11/02 (Paper No. 80) and page 13 of the Reply Brief, filed 8/9/02 (Paper No. 82).

Dasch et al. (U.S. Patent No. 5,772,998) in view of Ruoslahti et al. (U.S. Patent No. 5,583,103) AND/OR Bassols et al. (J. Biol. Chem. 263: 3039-3045, 1988) are taught above.

Dasch et al. (U.S. Patent No. 5,772,998) in view of Bassols et al. (J. Biol. Chem. 263: 3039-3045, 1988) and Raghu et al. (Am Rev Respir Dis 131: 281-29 (1985) are taught above.

In arguments to support the applicant's assertions to support the Rule 131 Declaration and accompanying Exhibits to antedate U.S. Patent No. 5,772,998 to Dasch et al., applicant has stated that given a generic method of decreasing TGF-β-induced production and deleterious accumulation of extracellular matrix associated with a pathology or a condition would render the specific fibrotic disorders such as glomerulonephritis, ARDS, cirrhosis or the liver and scarring as obvious species of one another (see Brief on Appeal, particularly Regarding Species Claims 23 and 25 and Regarding Genus Claim 21 on pages 18-24 of the Brief on Appeal, filed 3/11/02 (Paper No. 80) and page 13 of the Reply Brief, filed 8/9/02 (Paper No. 82).

Therefore, applicant's admissions are consistent with the prior art that one of ordinary skill in the art at the time the invention was made would have been motivated to treat glomerulonephritis and ARDS, given the teachings of Dasch et al. that TGF-β-specific antibodies could neutralize the effects of TGF-β in several disorders, including fibrotic disorders.

As indicated above, given the teachings of Dasch et al. that TGF-β-specific antibodies could neutralize the effects of TGF-β in a several disorders; one of ordinary skill in the art at the time the invention was made would have motivated to apply such TGF-β-specific antibodies in other disorders where TGF-β-played a role such as glomerulonephritis, as taught and indicated by Ruoslahti et al. and Bassols et al. Bassols et al. teach TGF-β regulates the expression of the extracellular matrix chondroitin/dermatan sulfate proteoglycans (see entire document, including Abstract, pages 3041 and 3043), including that TGF-β regulates proteoglycans in lung epithelial cell and fibroblast proliferation (see pages 3040-3041).

As indicated above, one of ordinary skill in the art at the time the invention was made would have motivated to apply such TGF-β-specific antibodies in other fibrotic disorders, such as interstitial lung fibrosis (see Dasch et al.) and ARDS, wherein interstitial fibrosis is a characteristic feature (see Raghu et al.). Similarly, given the role of the extracellular matrix in interstitial lung fibrosis and ARDS (see Dasch et al. and Raghu et al.) and the ability of TGF-β to stimulate lung epithelial cell and fibroblast proliferation, one of ordinary skill at the time the invention was made would have had a reasonable expectation that neutralizing TGF-β with TGF-β- specific antibodies would be effective in treating fibrotic diseases, including ARDS.

From the teachings of the references, it was apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was prima facie obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary

8. The following is a reiteration of the response to applicant's arguments of record as set forth in the Examiner's Answer, mailed 6/4/02 (Paper No. 81), as it applies to the current rejections under 35 U.S.C. § 103(a).

Applicant's arguments of record have been fully considered but are not found persuasive essentially for the reasons of record.

Applicant has maintained that the Border/Ruoslahti declarations under 37 C.F.R. § 1.131 sufficiently show that applicant's prior invention antedates the effective filing date of Dasch et al.

Applicant has acknowledged that Dasch et al. describe methods of neutralizing the inhibitory effects of TGF-β and several species of pathologies, including interstitial lung fibrosis, liver cirrhosis, fibrotic skin disorders such as scleroderma and scarring. In addition to the disclosure of Dasch et al., it has been noted that Dasch et al. claims methods of neutralizing the inhibitory effects of TGF-β with TGF-β-specific antibodies

Applicant has relied upon the averment by Border and Ruoslahti, pursuant to 37 C.F.R. § 1.131, that they conceived, prior to December 22, 1988, the claimed methods of decreasing TGF-β-induced production and deleterious accumulation of extracellular matrix associated with a pathology or a condition, including glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the liver and scarring), by contacting the affected tissued with anti- TGF-β antibody.

In response to the lack of supporting evidence or exhibits to show conception of all claim elements, applicant has asserted that the controlling case law indicated that not every claim element needs to be supported by accompanying Exhibits, provided that any missing element is supported by the Declaration itself.

Applicant has asserted that there exists no requirement to produce additional exhibits as appellant's reliance on the averments set forth in the Rule 131 declaration itself is entirely appropriate to establish conception of the invention prior to the effective date of the references. See Ex parte Ovshinsky, 10 USPQ2d (Bd. Pat. App. & Inter. 1989).

Applicant has acknowledged that the MPEP states that the evidence in the form may accompany the declaration, but does not require such extrinsic evidence. See MPEP 715.07.

With respect to Ex parte Swaney, 89 USPQ 618 (Bd. Pa. App. & Int. 1951), applicant has asserted that the conformity with the Swaney fact pattern has not been articulated by any court in the country.

Applicant has maintained that no court has held that in order to show conception prior to a critical date, an applicant has to provide one or more exhibits that explicitly or implicitly contain all elements of the claimed invention.

Further, applicant has argued that the declaration under 37 C.F.R. § 1.131 as well as a corroborating third party Declaration under 37 C.F.R. § 1.132, filed 3/15/01 (Paper No. 69), contains additional averments with regard to the claimed species with regard to conception of the claimed methods prior to December 22, 1988 or appellants' due diligence in pursuing reduction to practice of the claimed methods during the critical period. See Exhibits A-E.

Applicant has asserted that applicant's statements have been corroborated by Languino's (Exhibit A) averment that they conceived, prior to December 22, 1988, the claimed methods of decreasing the TGF-β-induced production and deleterious accumulation of extracellular matrix associated with a pathology or a condition by contacting the affected tissue with an anti-TGF-β antibody.

Applicant has relied upon the corroboration by Languino, who has stated that the during the time period Border conducted research related to the above-identified patent application in the same laboratory that the stated goal of using anti-TGF-β antibodies to inhibit TGF-β in order to decrease the deleterious TGF-β-induced production and accumulation of extracellular matrix associated with a disease, including kidney disease.

It has been acknowledged the current Border/Ruoslahti declaration under 37 C.F.R. § 1.131 and Languino declaration under 37 C.F.R. § 1.132 set forth that the stated goal of preparing anti-TGF-β antibodies was for their use to inhibit TGF-β in order to decrease deleterious TGF-β-induced production and accumulation of extracellular matrix associated with a pathology or condition, including glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the lung and scarring).

Applicant has relied upon that the Rule 131 Declaration itself that the stated goal of preparing anti-TGF-β antibodies was for their use to inhibit TGF-β in order to decrease deleterious TGF-β-induced production and accumulation of extracellular matrix associated with a pathology or condition, including glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the lung and scarring).

Other than the Rule 131 and 132 Declarations, neither applicant or Languino have provided any corroborating factual evidence to support the "stated goals" using anti-TGF-β antibodies to inhibit TGF-β in order to decrease the deleterious TGF-β-induced production and accumulation of extracellular matrix associated with a disease, including glomerulonephritis, adult respiratory distress syndrome, cirrhosis of the liver and scarring, by contacting the affected tissued with anti- TGF-β antibody.

Applicant has relied upon the "Animal Usage Form" (the redacted date of which is prior to December 22, 1998), which relates to the project entitled "Anti-human TGF-β Cyclic Peptide", which lists applicant Border and (non-co-inventor) Languino (but does not list not co-inventor Ruoslahti) as the investigators.

It is noted that Exhibit B to applicant's Rule 131 Declaration consists of laboratory notebook pages from (non-co-inventor) Languino's notebook to show the protocol of developing rabbit anti-TGF-β antiserum. The animals were for bled for anti-TGF-β antiserum December 13, 16 and 21 of 1988.

It is noted that the priority date of Dasch et al. (U.S. Patent No. 5,772,998) is December 22, 1988, which is one day after the final bleeding of the rabbits in the protocol of developing rabbit anti-TGF-β antiserum. Therefore, it appears that the rabbit anti-TGF-β antiserum was not tested prior to the effective priority date of Dasch et al. (U.S. Patent No. 5,772,998).

Also, applicant has relied upon Exhibit C, which is a conference abstract published for the Meeting of the American Society of Nephrology in San Antonio, Texas, which took place from December 11-14, 1988. This abstract is entitled "Transforming Growth Factor β (TGF β) Uniquely Regulates Production of Glomerular Extracellular Matrix". Applicant has asserted that this abstract is consistent with applicant's conception of treating pathologies related to TGFβ-mediated accumulation of extracellular matrix prior to December 22,1988. Applicant has submitted that because this abstract was presented to clinician attendees of the Nephrology meeting, given Drs. Border and Ruoslahti's medical training, such presentation was in the context of methods of suppressing the deleterious accumulation of TGF-β-induced extracellular matrix and not aimed merely at fulfilling the clinician's attendees' academic curiosity.

Scientific meetings serve a number of purposes including research interests as well as clinical studies for academic and company scientists and physicians

Applicant has asserted without evidence that based upon an in vitro experimental study disclosing the unique role of TGFβ in a kidney cell culture, the ordinary artisan would have extrapolated this finding to reducing extracellular matrix in a wide variety of distinct diseases with TGFβ-specific antibodies.

Given the disclosed uniqueness of TGFβ in a kidney cell culture, it is not readily apparent that the ordinary artisan would extrapolate the role of TGFβ broadly in all instances of the accumulation of extracellular matrix, given the contribution of a variety of factors to distinct conditions and pathologies. Also, the abstract does not mention or discuss methods of inhibiting extracellular matrix accumulation in glomerulonephritis nor the use of TGFβ-specific antibodies as an antagonist either in the context of kidney disease or broadly on any other condition involving the accumulation of extracellular matrix.

Again, applicant has asserted that Border and Ruoslahti have declared in their Rule 131 Declaration that at the time this abstract was submitted, they had already conceived of using anti-TGF-β antibodies was for their use to inhibit TGF-β in order to decrease deleterious TGF-β-induced production and accumulation of extracellular matrix associated with a pathology or condition, including glomerulonephritis.

Applicant has asserted that Exhibit D, a grant proposal laying out experimental aims, and Exhibit E, a publication of results obtained by performing experiments proposed in the grant proposal, speak to appellant's diligence in pursuing the reduction to practice of the claimed methods during the critical period.

Applicant has submitted that Exhibits D and E must be viewed in context, with Exhibit D being a grant proposal laying out experimental aims and Exhibit E, seven months later, a publication of results obtained by performing experiments proposed in the grant proposal.

Applicant has relied upon the excerpt form the Grant Proposal section entitled Specific Aims "to develop regiments for therapeutic intervention in the disease model by antibodies and other agents capable of neutralizing the TGF-β effect. In addition, the Experimental Design and Methods section states the proposal of several experiments "to block or ameliorate the action of TGF-β in the animal model of mesangial injury ... It is conceivable that one or more of these agents could be administered to the animal and/or infused directly into the kidney as therapeutic agents to prevent the expansion of mesangial matrix ... We expect that one or more of the agents to be tested will block the action of TGF-β. This information would be immediately applicable to the design of a study to treat humans with glomerulonephritis".

Applicant has relied upon Exhibit D corroborates appellant's averments that the reduction to practice of the claimed therapeutic methods were being diligently pursued from prior to December 22, 1988 until the filing date of the priority application and is consistent with appellant's averments in the Rule 131 Declaration.

Applicant also has relied upon Exhibit E which are excerpts of an updated draft manuscript, entitled "An Antiserum Against Transforming Growth Factor β Suppresses Experimental Glomerulonephritis" as it existed on August of 1989, which details the experiments proposed in the grant proposal. Applicant has submitted that the manuscript states that the results achieved in the experimental results with anti-TGF-β treatment warrant the expectation of similar benefits for treatment of human glomerulonephritis with other fibrosis-related diseases.

Here, applicant has asserted that the Exhibit E provides documentation that during the critical period appellant were diligently pursuing the reduction to practice of the claimed methods as averred in the Rule 131 Declaration.

Again, it is noted that the priority date of Dasch et al. (U.S. Patent No. 5,772,998) is December 22, 1988, which precedes the dates of both Exhibits D and E.

With respect to species - genus issues, it has been acknowledged that where the claims under rejection recite a species and the reference discloses the claimed species, the rejection can be overcome under 37 CFR 1.131 directly by showing prior completion of the claimed species or indirectly by a showing that the claimed species would have been an obvious modification of the species completed by applicant. See In re Spiller 182 USPQ 614 (CCPA 1974).

Applicant has acknowledged that Dasch et al. describes the two species of liver cirrhosis and scarring previously claimed but not adult respiratory distress syndrome (previously and currently claimed). Here, applicant has submit that the species is not disclosed in the Dasch et al. patent and therefore is not anticipated by Dasch et al. The current rejection with respect to the species ARDS is under 35 USC 103(a) and not under 35 USC 102(e).

Applicant has submitted that the Rule 131 Declaration itself shows prior invention of the species currently recited. Here, applicant has averred to the conception prior to December 22, 1988, and subsequent diligent reduction to production and deleterious accumulation of extracellular matrix associated with a pathology or a condition, including glomerulonephritis, adult respiratory distress syndrome, cirrhosis of the liver and scaring by contacting the affected tissued with anti TGF-β antibody. Further, applicant has argued that applicant's averments are supported by the Rule 132 Languino Declaration (Exhibit A) and the conference abstract (Exhibit C).

Here, applicant has asserted the controlling legal standard articulated by the Ovshinsky Court that it is entirely appropriate for appellant to rely on the averment set forth in the Rule 131 declaration themselves to establish conception of the invention prior to the effective date of the reference.

Applicant has submitted that in order to antedate a reference that has been cited against an application, distinct requirements exist that depend, in part, on whether the application claims a genus or species, and, in part, on whether the species are disclosed in the cited references.

Regarding previous genus claim 21, applicant argued that the issue is whether the species described by Dasch et al. would have been obvious to one of ordinary skill in the art in view of what the applicant's Rule 131 Declarations proves was completed with respect to the invention prior to the effective date of the reference.

Relying upon In re Clarke, 148 USPQ 665 (CCPA 1966), applicant submitted that one consideration is this regard is whether it can be shown that applicant had already appreciated that the invention was generic in nature prior to the reference date.

Applicant argued that the Rule 131 Declaration itself shows appellant's appreciation of the generic applicability of their invention to those pathologies and conditions associated with TGF-β-induced production and deleterious accumulation of extracellular matrix in a tissue.

Again, applicant relied upon the averments made in the Rule 131 Declaration and in the Languino 132 Declaration as well as the conference abstract.

Applicant has maintained that at the time the abstract was submitted, appellant already had conceived of using anti TGF-β antibodies in order to decrease deleterious TGF-β-induced production and accumulation of extracellular matrix associated with glomerulonephritis or other pathologies associated with TGF-β-induced expansion of the extracellular matrix.

With respect to the two conditions not explicitly described in the specification but mentioned in Dasch et al., scleroderma and interstitial lung fibrosis, applicant submitted that these conditions are obvious in view of what has been conceived prior to December 22, 1988 by appellant. Again, applicant had relied upon the averments in the Rule 131 Declaration. In addition, applicant stated that Dasch et al. discloses that scleroderma, like scarring, was known to be a fibrotic disease of the skin and that interstitial lung fibrosis, liked Adult Respiratory Distress Syndrome was known to be fibrotic disorder of the lung. Applicant submitted that given their prior conception of a generic method of decreasing the TGF-β-induced production of and deleterious accumulation of extracellular matrix associated with a pathology or a condition, applicant possessed so much of the invention as to encompass the Dasch et al. patent.

Regarding previous Species Claims 23 and 25 (now claims 21 and 35), applicant asserted that in order to antedate a reference that has been cited against an application, distinct requirements exist that depend, in part, on whether the application claims a genus or species and, in part, on the species disclosed in the cited references. Consequently, species claims must be separately examined with regard to whether applicant have made the necessary showing for prior invention of the claimed subject matter.

It has been noted that Rule 131 Declaration itself does not discuss the obviousness of species. For example, the Rule 131 Declaration does not state that scleroderma, like scarring, was known to be a fibrotic disease of the skin and that interstitial lung fibrosis, liked Adult Respiratory Distress Syndrome was known to be fibrotic disorder of the lung.

For the reasons set forth herein and of record, applicant submitted that the Rule 131 Declaration of March 15, 2001 is sufficient to antedate Dasch et al. the rejections under 35 USC 102(e) and 103(a), which would be applicable to the current rejection under 35 USC 103(a).

In addition to the above-mentioned, the following rebuttal has been provided.

While applicant has provided evidence / Exhibits to indicate prior conception to the prior art as it would read on Dasch et al. (U.S. Patent No. 5,772,998), applicant has not provided sufficient objective evidence to establish acts in this country commensurate in scope with the claimed invention, including as it would read upon targeting the current limitations of treating glomerulonephritis and ARDS.

With respect to current claim 21, it has been noted that the priority date of Dasch et al. (U.S. Patent No. 5,772,998) is December 22, 1988 and that the priority dates of Ruoslahti et al. (U.S. Patent No. 5,583,103) and Bassols et al. (J.Biol. Chem. 263: 3039-3045, 1988) are more than one year prior to applicant's priority date.

Similarly, with respect to current claim 35, it is noted that the priority date of Dasch et al. (U.S. Patent No. 5,772,998) is December 22, 1988 and that the priority dates of Bassols et al. (J. Biol. Chem. 263: 3039-3045, 1988) and Raghu et al. (Am Rev Respir Dis 131: 281-29 (1985) are more than one year prior to applicant's priority date.

The statements of stated goals in the Rule 131 Declaration and the Languino 132 Declaration appear to be the only sources for the "stated goals" of reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including glomerulonephritis, adult respiratory distress syndrome, cirrhosis of the liver and scaring by contacting the affected tissued with anti-TGF-β antibody.

Other than the Rule 131 and 132 Declarations, neither applicant or Languino have provided sufficient corroborating factual evidence to support the "stated goals" using anti-TGF-β antibodies to inhibit TGF-β in order to decrease the deleterious TGF-β-induced production and accumulation of extracellular matrix associated with a disease, including glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the liver and scarring), by contacting the affected tissued with anti- TGF-β antibody.

The affidavit or declaration must state FACTS and produce such documentary evidence and exhibit in support thereof as are available to shoe conception and completion of invention in this country, at least conception being at a date prior to the effective date of the references. See MPEP 715.07 and 715.07(c).

A general allegation that the invention was completed prior to the date of the reference is not sufficient. Ex parte Saunders, 1883 C.D. 23, 23 O.G. 1224 (Comm'r Pat. 1883). Similarly, a declaration by the inventor to the effect that his or her invention was conceived or reduced to practice prior to the reference date, without a statement of facts demonstrating the correctness of this conclusion, is insufficient to satisfy 37 CFR 1.131. See MPEP 715.07.

37 CFR 1.131(b) requires that original exhibits of drawings or records, or photocopies thereof, accompany and form part of the affidavit or declaration or their absence satisfactorily explained. See MPEP 715.07.

Applicant's reliance on "stated goals" in the Rule 131 and 132 Declarations is critical in supporting the generic claims as well as the claimed species. However, there is insufficient corroborating objective evidence that provides for using anti-TGF-β antibodies to inhibit TGF-β in order to decrease the deleterious TGF-β-induced production and accumulation of extracellular matrix associated with a disease, including glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the liver and scarring), by contacting the affected tissued with anti- TGF-β antibody. These are mere statements that do not provide a clear information the nature or time of the discussing the stated goals. For example, applicant's reliance on the other evidence never mention adult respiratory distress syndrome (nor cirrhosis of the liver and scarring). If the stated goals were intended to be broad, one would reasonably expect that these other pathologies and conditions would have been mentioned in the supporting evidence / Exhibits, such as grant proposals and manuscripts.

There is insufficient objective corroborating evidence to support the "stated goals", as asserted in the Rule 131 and 132 Declarations.

For example, the Languino was asked to assist in preparing anti TGF-β antibodies to inhibit TGF-β in order to decrease the deleterious TGF-β-induced production and accumulation of extracelluar matrix associated with a disease, including kidney disease. See Languino 132 Declaration (Exhibit A).

Applicant in conjunction with Languino rely upon Animal Usage Form, wherein the Project Goals are to produce quantities of anti-human TGF-β cyclized peptide for use in kidney disease research, wherein rabbits were immunized to produce high quality antiserum which can be used for identification of TGF-β in tissue samples and in vitro assays to study progression of kidney injury. See Animal Usage Form (Exhibit B).

Therefore, Animal Usage Form provides for generating anti -TGF-β antibody for in vitro kidney disease research. There does not appear sufficient direction or recognition that these in vitro research studies would lead to reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti TGF-β antibody.

The Languino 132 Declaration does not appear to provide sufficient direction or recognition that the in vitro research studies cited in the Animal Usage Form would lead to reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including glomerulonephritis, adult respiratory distress syndrome, cirrhosis of the liver and scaring by contacting the affected tissued with anti TGF-β antibody.

The laboratory notebook pages from Languino's notebook set forth protocols for immunizing rabbits with TGF-β. See Exhibit B.

Similar to the Animal Usage Form and the Languino 132 Declaration, the laboratory notebook pages do not appear to provide sufficient direction or recognition that the immunization procedures nor the in vitro research studies cited in the Animal Usage Form would lead to reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti TGF-β antibody. Although the in vitro research studies may have indicated the role of TGF-β in extracellular matrix accumulation in kidney cells, there appears insufficient evidence that one of ordinary skill in the art would have necessarily extrapolated that the production of producing a rabbit antiserum for such in vitro research studies would have led to the use of therapeutic antibodies in the treatment of human diseases and conditions, including glomerulonephritis and ARDS.

With respect to the conference abstract entitled "TGF-β is unique among growth factors in its metabolic effect on glomerular ECM" that the release of TGF-β could stimulate the expansion of ECM and progression to glomerulosclerosis. See Abstract of December 11-14, 1988.

Here, applicant has asserted that given their medical training as physicians, they already conceived of using anti TGF-β antibodies in order to decrease deleterious TGF-β-induced production and accumulation of extracellular matrix associated with other pathologies associated with TGF-β-induced expansion of extracellular cell matrix.

Here again, there is no statement in the conference abstract about the scope of pathologies or conditions, including adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti TGF-β antibody.

With respect to glomerulonephritis, the conference abstract discloses that TGF-β is unique among growth factors in its metabolic effects on glomerular extracellular matrix. The release of a substance like TGF-β in glomerulonephritis could stimulate the expansion of extracellular matrix and mediate the progression to glomerulosclerosis.

However, the conference abstract does not disclose treating or reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti -TGF-β antibody. Rather, this conference abstract is drawn studying the role of growth factors such as TGF-β in the progression of glomerulonephritis and does not disclose treating glomerulonephritis with anti -TGF-β antibody.

With respect to therapy, the Grant Proposal discloses the production of neutralizing antisera and it is conceivable that one or more of these agents could be administered to the animals and/or infused directly into the kidney as therapeutic agents to prevent the expansion of the mesangial matrix. See Grant Proposal, Background and Significance, Section B(e); see Exhibit D. The Grant Proposal expects that one or more of the agents to be tested will block the action of TGF-β, wherein the information would be applicable to the design of a study to treat humans with glomerulonephritis. The objective of the proposed studies was to test the hypothesis that growth factors such as TGF-β regulates the production and accumulation of extracellular cellular matrix in glomerular disease (see the last sentence of Section A Specific Aims).

Therefore, the Grant Proposal (January 1989) which was subsequent to the December 22, 1998 priority date of Dasch et al. (U.S. Patent No. 5,772,998) and is directed to testing the hypothesis relating to the role of TGF-β in regulating the production and accumulation of extracellular matrix in glomerular disease in order to design treatments for humans.

The Grant Proposal does not disclose treating reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti TGF-β antibody.

Applicant has relied upon excerpts of an undated draft manuscript (August 1989) entitled "An Antiserum Against Transforming Growth Factor β Suppresses Experimental Glomerulonephritis", which contains the in vivo protocol corresponding to Example VII of the instant specification. Again, this draft manuscript was subsequent to the December 22, 1998 priority date of Dasch et al. (U.S. Patent No. 5,772,998.

Here, the results contribute to the understanding of the pathogenesis of experimental nephritis and suggest a new form of therapy for glomerulonephritis with anti-TGF-β antibody (see Abstract and page 5, paragraph 2). Further, page 5, paragraph 2 of the manuscript discloses encouragement one to expect similar potential benefits in human glomerulonephritis and perhaps in other disease as well were fibrosis is a factor.

The draft manuscript discloses potential benefits of treatment of human glomerulonephritis with an anti-TGF-β antibody and does not disclose the scope of pathologies or conditions, including adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti TGF-β antibody.

Both the Grant Proposal and the draft manuscript are directly only at glomerulonephritis and testing the hypothesis of treating glomerulonephritis with an agent such as anti-TGF-β antibody.

Other than the Rule 131 Declaration, applicant has not provided any objective evidence to support prior conception, diligence and reduction of practice of reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti TGF-β antibody.

Further, with respect to the evidence that precede the December 22,1988 priority date of Dasch et al.; there is insufficient objective evidence that the ordinary artisan would recognize the reliance on the Animal Usage Form, where the Project Goals of producing anti-human TGF-β cyclized peptide for use in kidney disease research (Animal Usage Form; Exhibit B) and on the conference abstract, which discloses that TGF-β is unique among growth factors in its metabolic effects on glomerular extracellular matrix would read on methods of reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti TGF-β antibody.

In contrast to applicant's reliance on Languino's statement of "associated with a disease, including kidney disease" (Languino 132 Declaration), "associated with a disease" does not provide sufficient direction nor support for methods of reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti TGF-β antibody.

Diseases and conditions such as the claimed glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) as well as the interstitial lung fibrosis and scleroderma as disclosed by Dasch et al. are distinct because the pathological conditions differ in etiologies and therapeutic endpoints. For example, these diseases and conditions encompass different tissues and organs as well as etiologies and therapeutic endpoints.

Applicant has not provided objective evidence that the ordinary artisan would have recognized that the production of TGF-β antibody and in vitro assays on the role of TGF-β in mesangial cultures conducted by appellant prior to December 22,1988 as well as the abstract, grant proposal and draft manuscript on determining the contribution of TGF-β in experimental nephritis would read broadly on diseases and conditions with varying etiologies and therapeutic endpoints, such as the claimed glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis and scarring).

Applicant has relied upon In re Ovshinsky to indicate that exhibits accompanying the Rule 131 Declaration need not support all of the claimed limitations inasmuch as missing feature may be supplied by the declaration itself. In the instant application, the only support for methods of reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti- TGF-β antibody is the asserted "stated goals" in the Rule 131 Declaration. There is insufficient corroborating objective evidence prior to nor subsequent to the December 22, 1988 priority date of Dasch et al. to support the claimed diseases of adult respiratory distress syndrome (as well as cirrhosis of the liver and scarring) nor that these diseases were obvious modifications of testing the role of TGF-β in experimental in vitro mesangial cultures or in vivo murine nephritis models. There is insufficient corroborating evidence to support the asserted " stated goals" in the Rule 131 Declaration to encompass claimed diseases of adult respiratory distress syndrome (as well as cirrhosis of the liver and scarring).

Even with respect to treating glomerulonephritis which was the focus of appellant's Exhibits, it is noted that the Animal Usage Form and the conference abstract that precede the December 22, 1988 priority date of Dasch et al. are limited to studies of determining the presence or role of TGF-β in mesangial cultures, as an in vitro model of glomerulonephritis and not to treatment of glomerulonephritis with anti-TGF-β antibody. While the role of TGF-β in mesangial cultures was tested prior to December 22, 1988, the use of anti-TGF-β antibody in experimental in vitro mesangial cultures or in vivo murine nephritis models had not been conducted prior to December 22, 1988. Border's Grant Proposal as well as draft manuscript are couched in terms of potential benefits to treating human glomerulonephritis with some agents.

Again, with respect to filing declarations under 37 CFR 1.131, the showing of facts shall be such, in character and weight, as to establish reduction to practice prior to the effective date of the reference, or conception of the invention prior to the effective date of the reference coupled with due diligence from said date to a subsequent reduction to practice or to the filing of the application. Original exhibits or drawings or records or photocopies thereof must accompany and form part of the affidavit or declaration or their absence satisfactory explained. See MPEP 715.07.

While applicant has asserted that exhibits and declarations are not required, Rule 131 clearly requires supporting documentation to the Rule 131 Declaration or a satisfactory explanation to explain their absence.

Applicant has not provided a sufficient explanation as to why their is not supporting evidence for the stated goals for methods of reducing the deleterious accumulation of extracellular matrix associated with the scope of pathologies or conditions, including glomerulonephritis and adult respiratory distress syndrome (as well as cirrhosis of the liver and scaring) by contacting the affected tissued with anti-TGF-β antibody.

Further, the evidentiary evidence prior to December 22, 1988 appears drawn to determining the contribution of TGF-β to extracellular matrix accumulation in mesangial cultures as a model of glomerulonephritis and not necessarily to the treatment of glomerulonephritis with anti-TGF-β antibody or broadly to the inhibition of extracellular matrix in various pathologies and conditions(e.g. ARDS).

Subsequent to December 22, 1988, the Border Grant Proposal and co-authored draft manuscript appear to draw on experimental in vivo models to test agents that may be useful in treating glomerulonephritis. Again, these documents do not mention the inhibition of extracellular matrix in various pathologies and conditions such as adult respiratory distress syndrome (as well as cirrhosis and scarring).

With respect to applicant's arguments on genus - species claim limitations, it has been noted that previous generic claim 21 recites "a method of decreasing the deleterious accumulation of extracellular matrix associated with a pathology or a condition wherein TGF-β-induced production and deleterious accumulation of extracellular matrix comprising contacting the tissue with an anti-TGF-β antibody that bind to TGF-β".

Applicant was not in possession of the generic invention prior to the effective date or activity of the prior art. The Rule 131 Declaration and corroborating evidence does not provide the minimum disclosure required for the given scope of "pathologies and conditions" to antedate the prior art. A reference or activity which discloses several species of a claimed genus can be overcome directly under 37 CFR 1.131 only by a showing the applicant completed, prior to the date of the reference or activity, all of the species shown in the reference. In re Stempel, 113 USPQ77 (CCPA 1957). See MPEP 715.03(B).

Given the scope as well as the previous recitation of "pathology and condition" recited in the independent claim as well as the dependent claims, the claims have not been simply directed toward suppressing the activity of "the deleterious accumulation of TGF-β-induced extracellular matrix in the tissue". Rather, the recitation of "pathology and condition" clearly indicates the context of multiple diseases or condition and determining which ones (or at least a representative number of species) are relevant or appropriate to the ordinary artisan.

Again, both applicant and examiner have prosecuted the claims as if they stood or fell together. The only issue of record has been whether appellants' Declaration under 37 C.F.R. 1.131, filed on March 15, 2001 was sufficient to antedate U.S. Patent No. 5,772,998. Applicant did not appear to distinguish the genus and species claims prior to this Appeal Brief.

While applicant has provided some documentary support as evidence of conception, diligence and reduction to practice; these documents or exhibits together with the comments in the Border/Ruoslahti declaration are not clear on their face as they read on conception, diligence and reduction to practice commensurate in scope with the claimed methods in order to antedate the prior art. Appellant has the burden to explain the contents of the pages as proof of acts amounting to conception, diligence and reduction to practice. See In re Borkowski and Van Venrooy 184 USPQ 29 (CCPA 1974). Absent a clear explanation of pointing out exactly what facts were established and when they were established and relied upon by applicant, the Rule 131 and 132 Declarations and Exhibits provide insufficient assistance in enabling the PTO to determine applicant's assertions of conception, diligence and reduction to practice before the prior art as it reads on methods of decreasing the deleterious accumulation of extracellular matrix associated with pathologies and conditions, including glomerulonephritis and ARDS previous to the effective dates of the prior art references.

9. In accordance with MPEP 2308.01, the following is set forth.

The application of the Dasch et al. (U.S. Patent No. 5,772,998) (in view of Ruoslahti et al. (U.S. Patent No. 5,583,103) AND/OR Bassols et al. (J. Biol. Chem. 263: 3039-3045, (1988) OR in view of Bassols et al. (J. Biol. Chem. 263: 3039-3045, (1988) and Raghu et al. (Am Rev Respir Dis 131: 281-29 (1985) are taught above) cannot be overcome by an affidavit or delcaration under 37 CFR 1.131 but only through interference proceedings. An affidavit under 37 CFR 1.608(b) or evidence and an explanation under 37 CFR 1.9608(b), as appropriate, must be submitted and it should be stated, if applicable, that patentee has been accorded the benefit of an earlier U.S. application.

It is noted that the Board of Patent Appeals and Interferences' Decision on Appeal NO.: 2002-2285, mailed 12/19/02 (Paper No. 85), states: "the issue of priority of invention cannot be resolved through use of a declaration under 37 CFR 1.131 but, rather, only through an interference proceeding (see page 4, paragraph 1).

10. The non-statutory double patenting rejection, whether of the obvious-type or non-obvious-type, is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent. *In re Thorington,* 418 F.2d 528, 163 USPQ 644 (CCPA 1969); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); *In re Van Ornam*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); and *In re Goodman*, 29 USPQ2d 2010 (Fed. Cir. 1993).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321 (b) and (c) may be used to overcome an actual or provisional rejection based on a non-statutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.78 (d).

Effective January 1, 1994, a registered attorney or agent of record may sign a Terminal Disclaimer. A Terminal Disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

11. Claim 21 and 35 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-6, 8-17 and 25-29 of copending application USSN 08/3459,865 alone

or in view of Dasch et al. (U.S. Patent No. 5,772,998), Ruoslahti et al. (U.S. Patent No. 5,583,103), Bassols et al. (J. Biol. Chem. 263: 3039-3045, 1988) and well as Raghu et al. (Am Rev Respir Dis 131: 281-29 (1985) as well as

and in further evidence of applicant's admission that given a generic method of decreasing TGF-β-induced production and deleterious accumulation of extracellular matrix associated with a pathology or a condition would render the specific fibrotic disorders such as glomerulonephritis, ARDS, cirrhosis or the liver and scarring as obvious species of one another (see Brief on Appeal, particularly Regarding Species Claims 23 and 25 and Regarding Genus Claim 21 on pages 18-24 of the Brief on Appeal, filed 3/11/02 (Paper No. 80) and page 13 of the Reply Brief, filed 8/9/02 (Paper No. 82).

Although the conflicting claims are not identical, they are not patentably distinct from the present and copending claims are drawn to the same or nearly the same methods of treating fibrotic conditions or diseases with TGFβ-specific antibody. Given the recitation of the present / pending as well as the teachings set forth above, one of ordinary skill in the art would have been motivated to treat a variety of fibrotic conditions or diseases, including those diseases and conditions of the instant / copending claims (e.g. glomerulonephritis, ARDS, scarring, cirrhosis) with TGFβ-specific antibody with an expectation of success at the time the invention was made.

This is a *provisional* obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

12. It is noted that USSNs 08/407,942 and 08/457,709 are not available to the examiner at this time. Therefore, a determination of obviousness-type double patenting could not made at this time. If these copending application recite methods of treating diseases, including glomerulonephritis / ARDS or methods of inhibiting extracellular matrix production / fibrosis with TGFβ-specific antibody , then these applications would be subject to a rejection under obviousness-type double patenting.

13. No claim is allowed.

14. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Phillip Gambel whose telephone number is (703) 308-3997. The examiner can normally be reached Monday through Thursday from 7:30 am to 6:00 pm. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christina Chan can be reached on (703) 308-3973. Any inquiry of a general nature or relating to the status of this application should be directed to the Technology Center 1600 receptionist whose telephone number is (703) 308-0196.

Papers related to this application may be submitted to Technology Center 1600 by facsimile transmission. Papers should be faxed to Technology Center 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center telephone number is (703) 872-9306.

Phillip Gambel, PhD.
Primary Examiner
Technology Center 1600
August 19, 2003